



Editorial

A Neuron Story

Would you believe that the diseases which inclined to the best known differentiated cell of the human are the result of a love story? Alzheimer disease (AD) with amyloid deposits in brain and with degenerating neurons; Huntington disease (HD) which polyglutamine expansion causes huntingtin protein to aggregate into inclusion bodies, which are thought to cause the neuronal death that follows. Parkinson disease (PD) is caused by progressive loss of dopaminergic neurons from the substantia nigra. The proximate cause of this loss is unknown. May be as a part of a same story.

Alzheimer's disease will hit over 10 million patients in Europe by 2025 with a major impact on health care systems and society at large. Investment in fundamental and applied research is imperative to understand its causes, etiology and to identify drug targets and development of active chemical compounds into drugs. Monitoring memory performance and brain pathology allows to define beneficial effects of novel and innovative therapies (4).

Pathologists have long used microscopes to observe disease-related changes, but it is not always clear if all changes are bad. In Huntington's disease, polyglutamine expansion causes huntingtin protein to aggregate into inclusion bodies, which are thought to cause the neuronal death that follows. A new robotic microscope has been used to study this system: once focused on a neuron it returns to that same neuron after any interval. Surprisingly, inclusion bodies did not cause neuronal death but reduced mutant huntingtin levels and improved survival, suggesting that they are a coping response (1).

By identifying pathogenic, incidental or beneficial change, diseases can be better understood and therapeutic targets identified. Restoration of dopamine levels attends only to the manifestatins of neurodegeneration in

Parkinson's Disease, not to the actual cause of the loss of dopaminergic neurons (2).

A neuronal network is formed by neurons from the same nucleus. It is described by the connections between these neurons, the type of synapse (glutamatergic, GABA ergic, etc.), and the arrangement of the synapses on somato-dendritic trees. Networks have in common a basic organization. There are always principal cells and most often interneurons (the subthalamic nucleus is, for example, devoid of interneurons whereas striatum contains many different types of these). Interneurons are always connected so as to provide feedforward and feedback inhibitions (3).

Neuroprotection may arise indirectly by stimulating an intermediate effector cell (eg. Astroglia) rather than directly by sustaining degenerating neurons. Improved understanding of the molecular mechanism of PD, AD, or HD will permit targeting of therapy to etiology rather than symptoms.

We can only enjoy by alerting neuronal network or in some way effective molecular therapies in order to conclude this neuron story to an happy-end.

Editor

Prof. Nezh Oktar M.D.

noktar@med.ege.edu.tr

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Comments and feedback:

E-mail: norolbil@med.ege.edu.tr

URL: <http://www.med.ege.edu.tr/norolbil>

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